Kidney International, Vol. 58 (2000), pp. 462–466

Hepatitis C virus in hemodialysis patients

To the Editor: We have read with interest the recent paper by Rampino et al [1]. The authors observed milder histological lesions in patients receiving hemodialysis (HD) treatment in comparison with control patients with no renal disease, suggesting that hepatocyte growth factor serum levels attained during dialysis would attenuate the liver damage caused by hepatitis C virus (HCV), concluding that HCV-related liver disease is more benign in HD patients. This interesting article deserves some comments. First, the route by which HCV infection was transmitted was probably different in the two groups. When transmission is patient-to-patient (the most frequent route in HD patients, at least since 1992), the volume of contaminant may be considerably less than in cases of transfusion-acquired infection. The size of the infecting dose of the virus may be a confounding factor, as inflammatory activity upon histological examination has been reported to be greater in cases of transfusion-acquired HCV infection. We agree with the authors that histological lesions of the liver in HD patients with HCV infection are mild. However, a histological study showed that 9.5% of anti-HCV positive HD patients had liver cirrhosis [2]. In our dialysis unit we have observed that 17.5% of anti-HCV positive long-term HD patients evolve to liver cirrhosis a median of 10 years after initiating renal replacement therapy. Furthermore, in HD patients [3]—though not in patients with normal renal function [4]—the presence of anti-HCV has been associated with a higher risk of mortality. HCV-related liver disease in HD patients shows particular characteristics: ALT levels are below those of the nonuremic population; the route of transmission is different; and histological lesions of the liver are mild. Nevertheless, in our opinion further studies with a long-term follow-up are necessary before concluding that this disease is more benign in HD patients than in patients with no renal disease.

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Reply from the authors

Drs. Espinosa, Martín-Malo and Aljama assume that the transmission of HCV infection was patient-to-patient in patients on hemodialysis, while it was transfusion-acquired in nonuremic patients. Thus, the different route of infection would account for the different course of liver disease. Though we did not analyze in detail the route of infection in single patients, we challenge the assumption that the prevalence of post-transfusional hepatitis was higher in patients without renal disease. In fact, in Italy testing for anti-HCV antibody in transfused blood has been mandatory since 1990, and in all our patients (both uremic and nonuremic) seroconversion occurred later than the first semester of 1994. Therefore, infection through an infected transfusion should have occurred at least 3.5 years before seroconversion, an unusually long latency time. In addition, as stated in the **Methods** section, patients without renal disease were recruited among otherwise healthy subjects undergoing periodical anti-HCV testing because of professional or social risk for infection. Thus, the population checked consisted mainly of health care workers who had contact (external or through needlestick) with HCV-positive blood and of spouses of infected people. Espinosa et al claim that a histological study [1] showed that 9.5% of the anti-HCV positive patients on hemodialysis had liver cirrhosis. Actually, in the cited study biopsy-proven cirrhosis was found only in 3 out of 65 anti-HCV patients, that is, in less than 5%. Pereira et al found a higher risk of mortality in patients referred for renal transplantation who were anti-HCV positive compared to anti-HCV negative patients [2]. However, survival was computed from the onset of treatment for end-